BOOK REVIEW

Transesophageal echocardiography: basic principles and clinical applications. A J Labovitz, A C Pearson. New York: Lea & Febiger, 1993. (Pp 157; £32). ISBN 0–8121–1578–3.
Transesophageal echocardiography: clinical and intraoperative applications. J Missri. London: Churchill Livingstone, 1993. (Pp 248; £110). ISBN 0–443–08852–7.
Transesophageal echocardiography: in clinical practice. G R Sutherland, J R T C Roelandt, A G Fraser, R H Anderson. London: Gower Medical Publishing, 1991. (Pp 213; £99). ISBN 0–397–44640–3.

The books on transoesophageal echocardiography by Labovitz and Pearson and by Missri were published this year whereas that by Sutherland and coauthors was published in 1991. All three books assume some knowledge of Doppler and echocardiographic principles and are aimed at those with some experience of transthoracic imaging.

Technical problems have until recently limited the application of transoesophageal echocardiography. Thus all three books provide a history of the recent technical developments that have enabled this technique to reach the clinical arena. In addition all three books detail what is required to develop a clinical transoesophageal echocardiography service though in practice this information is hardly sufficient.

The technique of transoesophageal Doppler and echocardiography has significant advantages over transthoracic imaging. The close proximity of the oesophagus and gastric fundus to the intrathoracic cardiac structures allows enhanced resolution and imaging of structures inaccessible to the transthoracic view, including the descending thoracic aorta and left atrial appendage. Accordingly a significant portion to all three books is devoted to 2D photographs, of both normal and abnormal cardiac structures, in monochrome and colour. Labovitz and Pearson's book contains the smallest number of figures at around 150, while Sutherland and coauthors include over 300. The quality of the figures is especially impressive in the books by Missri and Sutherland and coauthors. Sutherland in particular supplements the high quantity pictures with excellent legends, schematic diagrams, and postmortem photographs required to aid interpretation.

Labovitz and Pearson's book is the only one in softback and is much the smallest of the three. The authors aim to provide only a basic introduction to transoesophageal imaging with particular reference to a variety of relevant pathological states. Commonly encountered clinical states are covered but the content is not comprehensive, nor is it intended to be. Each chapter includes a bibliography but not references. The text is generally well written and the chapters well organised. However, the authors include chapters on "interventional TEE" and outline its use in "critically ill patients". This latter chapter includes a mismash of clinical states covered more appropriately and in more detail in the relevant chapters and adds little to the reader's knowledge of transoesophageal echocardiography. "Interventional TEE" is misleading as the chapter refers only to the use of transoesophageal imaging in relation to interventional procedures such as balloon dilatation of the mitral valve, closure of atrial and ventricular septal defects, and myocardial biopsy.

Missri and Sutherland and coauthors aim, with success, to provide more extensive, in depth and up to date reviews of transoesophageal Doppler and echocardiography with particular reference to its indications, values, and limitations. Both texts are very well organised, comprehensive, and informative and there is little to choose between them. Missri includes 18 separate chapters, and Sutherland and coauthors 15, each with appropriate references. The areas covered in detail include dysfunction of prosthetic and native valves, congenital heart disease, the evaluation of the source of cardiac emboli, as well as paediatric and intraoperative use. Missri also includes a chapter on future applications of TOE. Missri's chapter on prosthetic valve evaluation and recognition of dysfunction is typical of many in his book. Transoesophageal imaging is not without its limitations and these are well described with respect to assessing prosthetic valves. Missri then follows this with a detailed but concise description of the basic principles behind various prosthetic valves. Examples of both the normal and abnormal function of a variety of prosthetic valves are well illustrated by colour Doppler images.

Sutherland's book is the largest of the three. Its four main authors are supported by an impressive eleven contributors. As a result Sutherland and coauthors have written with at least the same detail as Missri on a similar range of subjects. Though it is two years older their book is not out of date. For example the chapter on pulmonary venous flow, which was extremely difficult to measure accurately until the advent of TOE and is currently the subject of active research, is particularly well covered. Sutherland details normal pulsed wave Doppler pulmonary venous flow and contrasts this with several illustrated examples of abnormal flow. Pulmonary venous flow, however, is of unproven clinical significance at present.

In summary, Labovitz and Pearson succeed in providing a good introduction to transoesophageal echocardiography principally for those with some experience in transthoracic imaging. The books by Missri and Sutherland and coauthors are much larger, better written, and more detailed. They provide a comprehensive review of all aspects of transoesophageal echocardiography. As up to date, in depth reference books those of both Missri and Sutherland *et al* can be strongly recommended, though neither is particularly cheap.

STEPHEN FORT

The titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the UK and for members of the British Forces Overseas, but overseas customers should add £2 per item for postage and packing. Payment can be made by cheque in sterling drawn on a UK bank, or by credit card (MasterCard, VISA, or American Express) stating card number, expiry date, and your full name.

LETTERS TO THE EDITOR

- *The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.*
- *All letters must be typed with double spacing and signed by all authors.*
- *No letter should be more than 600 words.*
- *In general, no letter should contain more than six references (also typed with double spacing).*

Emergency percutaneous transluminal coronary angioplasty (PTCA) for intractable ventricular arrhythmias associated with acute anterior myocardial infarction

SIR,—Fitzpatrick *et al* report the value of emergency PTCA in the setting of acute myocardial infarction complicated by life-threatening ventricular arrhythmias (*British Heart Journal* 1993;**69**:453–4). We were recently involved in the management of a similar case.

A 49 year old man was admitted as an emergency with a 1 hour history of chest pain. He was a non-smoker with a history of hypertension and a family history of coronary disease. The ECG showed typical changes of an acute anterior myocardial infarction with right bundle branch block. He was given aspirin and intravenous streptokinase (1·5 MU). Thirty minutes later he sustained ventricular fibrillation and cardiac arrest. Serum electrolyte concentrations were within normal limits. During the subsequent 30 minutes he required more than 20 DC countershocks for repeated ventricular fibrillation. There was no significant response to appropriate doses of intravenous lignocaine and amiodarone.

An intra-aortic balloon pump (IABP) was inserted and he was transferred to the cardiac catheterisation laboratory still requiring repeated DC countershocks. At cardiac catheterisation the proximal segment of the left anterior descending (LAD) artery was found to be occluded. The circumflex and dominant right coronary arteries were without significant disease. Left ventricular angiography was not performed. A guide wire was passed across the occlusion and 100 000 units of streptokinase was infused into the left system with lysis of the proximal LAD thrombus. A shallow ulcerated plaque was visualised at the site of the occluding thrombus. In addition, there was a significant stenosis in the mid LAD coronary artery. Immediately after anterograde flow was restored sustained sinus rhythm returned and further DC countershocks were not needed.

He was transferred to the intensive care unit where he was ventilated overnight. Within 24 hours he was weaned from the ventilator and the IABP was removed. Inotropic support was stopped the next day. Four days after admission radionuclide ventriculography showed considerable anteroseptal hypokinesia with an ejection fraction

of 35%. Repeat coronary angiography before discharge showed a patent LAD coronary artery.

Fitzpatrick *et al* suggest that the improvement in rhythm control and haemodynamic status seen after IABP insertion was due to spontaneous re-opening of the infarct-related vessel. Our observations confirm that patency of the infarct-related vessel may be associated with arrhythmia control. There have been no randomised trials of the value of emergency intervention in such circumstances. Nevertheless, it is clear that restoration of vessel patency by intracoronary thrombolysis, with or without angioplasty, may be a life-saving intervention in acute myocardial infarction complicated by ventricular arrhythmias that remain uncontrolled despite appropriate drug therapy

M HARGREAVES
K CHANNON
O ORMEROD
Cardiology Department, John Radcliffe Hospital, Headington, Oxford OX3 9DU

Lack of rebound during intermittent transdermal treatment with glyceryl trinitrate in patients with stable angina on background β blocker

SIR,—Holdright *et al* in presenting their evidence of lack of rebound during intermittent transdermal treatment with glyceryl trinitrate in patients with stable angina on background β blocker (*British Heart Journal* 1993;**69**:223-7) unfortunately left out one important limitation of their study. The rebound effect described in previous studies was seen in the exercise test in the morning after the patch had been removed the previous evening,[1] or as an increase in numbers of attacks in the evening after removal of the patch worn during the day.[2] In the daytime, patients generally spend their time upright and walking around, activities resulting in greater sympathetic activation and more hydrostatic pressure in the lower extremities than when the patch is worn at night when patients rest supine most of the time and are subject to low sympathetic activation. Parker *et al* showed that intermittent daytime patch administration of glyceryl trinitrate in young healthy volunteers was associated with increases in plasma catecholamines, plasma renin, and antidiuretic hormone.[3] Such a mechanism may also operate in elderly patients, particularly as increasing age seems to be related to increased sensitivity to glyceryl trinitrate.[4]

Though Holdright *et al's* explanation that background β blocker treatment was responsible for the absence of the rebound effect is quite plausible, it remains unproven until the same type of study has been performed with daytime application of the glyceryl trinitrate patch.

GUNNAR NYBERG
Department of Medicine, Östra Hospital, Göteborg and Clinical Pharmacology, Astra Hässle, Mölndal, Sweden

1 DeMots H, Glasser SP. Intermittent transdermal nitroglycerin therapy in the treatment of chronic stable angina. *J Am Coll Cardiol* 1989;**13**:786–93.
2 Ferratini M, Pirelli S, Merlini P, Silva P, Pollavani G. Intermittent transdermal nitroglycerin monotherapy in stable exercise-induced angina: a comparison with a continuous schedule. *Eur Heart J* 1989;**10**: 998–1002.
3 Parker JD, Farrel B, Fenton T, Conamin MA, Parker JO. Counter-regulatory responses to continuous and intermittent therapy with nitrolycerin. *Circulation* 1991;**84**:2336-45.
4 Cahalan MK, Hashimoto Y, Aizawa K. Verotta D. Ionescu P, Balea M, *et al.* Elderly, conscious patients have an accentuated hypotensive response to nitroglycerin. *Anesthesiology* 1992;77:646-55.

This letter was shown to the authors, who reply as follows:

SIR,—Dr Nyberg raises an interesting point about the mechanism of rebound associated with intermittent nitrate therapy. It is plausible that patch application at night resulted in less neurohumoral activation than would have occurred with daytime therapy. However, as we originally stated,[1] we based the study design on the known circadian pattern of angina in order to maximise the likelihood of detecting rebound after patch removal. Exercise tests were performed in the morning to coincide with the well-recognised morning peak of ischaemia. The benefits of such a schedule have to be weighed against the possibility that nocturnal patch application results in less sympathetic activity than daytime therapy. However, neurohumoral activation is only one mechanism that could be responsible for the rebound phenomenon. Other mechanisms that are independent of the timing of patch application include sulphydryl depletion, desensitisation of soluble guanylate cyclase, and plasma volume shifts related to altered capillary pressure.[2 3]

D R HOLDRIGHT
R J KATZ
C A WRIGHT
J L SPARROW
A K SULLIVAN
A D CUNNINGHAM
K M FOX
Royal Brompton National Heart and Lung Hospital, Sydney Street, London SW3 6NP

1 Holdright DR, Katz RJ, Wright CA, Sparrow JL, Sullivan AK, Cunningham AD, Fox KM. Lack of rebound during intermittent transdermal treatment with glyceryl trinitrate in patients with stable angina on background β blocker. *Br Heart J* 1993;**69**:223-7.
2 Needleman P, Johnson EM Jr. Mechanism of tolerance to organic nitrates. *J Pharmacol Exp Ther* 1987;**184**:7009–15.
3 Abrams J. Clinical aspects of nitrate tolerance. *Eur Heart J* 1991;**12**(Suppl E):42-52.

Is there such a thing as normal sinus rate?

SIR,—Although in his viewpoint Professor Meijler wrestled with the perpetually belaboured concept of "normal", he began: "The currently accepted limits for a normal (sic) sinus rate were set at 60 and 100 beats per minute by Kossmann in 1953".[1] These limits were set long before 1953 in consecutive editions of the New York Heart Association's Nomenclature and Criteria in 1928 for "regular sinus rhythm" and subsequently in the 4th (1943) edition and thereafter for "normal sinus rhythm"[2] at least partly because 60 beats per minute represents exactly five 200 ms boxes on ECG paper and 100 beats per minute represents three 200 ms boxes. Kossmann clearly described these limits as being chosen "for convenience and for uniformity of designation." In any event, in our paper we were not concerned with electrocardiography, but rather with clinical and epidemiological appropriateness.[3]

Professor Meijler referred to Murphy's seven definitions of normal.[4] In a reply to the single letter that was critical of our work, I have already cited Murphy and have emphasised that our proposal of "normal" was as an *operative* definition in Murphy's sense of "acceptable"[5] (not noted by Professor Meijler). My colleagues and I understood that under conditions other than resting daytime ones individuals could indeed have sinus heart rates that are normal though beyond both of the operational (resting) limits that we proposed, as, for example, during sleep or during the range of physical activity. Moreover, if our paper were regarded as a redefinition of sinus tachycardia and bradycardia, the word "normal" could have been omitted from the title with no loss of message.

Professor Meijler challenges our study group (500 patients) as perhaps not being "a sufficiently large and appropriately stratified healthy sample". However, as we reported, our results accord with the results in the 5000 patients reported by the Framingham Heart Study.[3] Moreover, a personal message from Professor Rauttaharju of EPICORE ((Cardiology) Epidemiology Coordinating and Research Centre) (Alberta, Canada) cites comparable results in over 18 000 normal subjects. He has designed an abstract (now accepted) for our joint presentation at a forthcoming scientific meeting.

On the basis of past contributions Professor Meijler's views deserve respectful attention. However, in a survey of 136 distinguished members of the American College of Cardiology (many of them Professor Meijler's peers) over 90% agreed with the operational rate limits of 50 to 90 beats per minute with only two votes for the status quo and with the remainder supporting different variants.[6]

Professor Meijler refers to the increased cardiovascular mortality predicted by increased resting heart rates and asks "How important is the difference in mortality between patients with heart rates of 90 and 100"? The answer awaits an appropriately designed and executed investigation. No formal investigation underlay the traditional 60 to 100 beats per minute range. Why then does Professor Meijler prefer this range to ours, which is based on the results of an appropriately designed study and are consistent with Framingham and EPICORE data? Indeed, so few subjects had rates between 90 and 100 beats per minute that there may, indeed, be a critical difference in that range.

In his last sentence Professor Meijler offers a truism—that is, trivial changes in "normal" boundaries irrespective of statistical significance may not reflect biological significance. Yet, his very first sentence about "accepted limits" tacitly agrees that there can be conventional ("accepted") normal limits. In Professor Meijler's hospital do reports on electrocardiograms (computer generated or other) use "normal sinus rhythm" for regular sinus rhythms between 60 and 100 beats per minute and "sinus tachycardia" and "sinus bradycardia" for faster and slower rates?

Terminology greatly influences thought patterns, because "linguistic usage shapes